12.

Congenital Macrofollicular Cystic Colloid Goiter in a Dromedary.

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(Plate I).

Congenital colloid goiter, especially with cystic change, is a rarity not only among the lower animals but in the human species as well (4,12). More infrequently is it a cause of intrauterine fetal death and of dystocia, as occurred in the following case at the Zoological Park.

Case.

An adult female dromedary, multiparous although without any record of previous pregnancies at the Zoological Park, went into labor following an uneventful full-term period of gestation. Parturition failed to proceed normally. A vaginal examination by Dr. Schroeder, Veterinarian of the New York Zoological Park, revealed that the fetus was in a dorso-sacral position with the hind legs retracted, so that the tarsi presented. In addition, early maceration was evident, indicating that the fetus was dead. A very large cystic mass was palpated on the neck of the fetus. This, together with the head and forelegs, was too large to pass through the birth canal. Embryotomy was performed.

Pathological Examination.

The head of the fetus was shortened in the antero-posterior diameter, being a so-called "bull-dog head." Except for this and the tumor of the neck there were no other congenital malformations.

The tumor consists of two equal and similar masses which occupy the normal positions of the thyroid lobes, and is, in fact, a markedly enlarged thyroid gland. Each lobe measures 25 centimeters in length, 15 centimeters in width, and 8 centimeters in thickness. (The length of a normal thyroid in an adult dromedary is 3-4 inches (7-10 centimeters) according to Leese (5)). The enlargement appears diffuse and uniform. A dense gray fibrous capsule to which is adherent several strands of muscle surrounds each lobe. The gland is soft in consistency and feels cystic on palpation.

The cut surface of the gland is moist, glistening, pale brownish in color, and gelatinous in appearance. It possesses a coarsely honeycombed structure, being composed of numerous bulging, round and angulated, very thin walled cysts. (Pl. I). These vary in size from about 0.5 to 2 centimeters in diameter, most of them being about 1 centimeter. The cystic spaces are all filled with translucent, pale brown colloid. In some areas extending in from the capsule there are broad gray fibrous septa. No normal thyroid structure is grossly observed.

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On microscopic examination moderate autolysis is noted. The capsule is seen to be composed of a broad band of dense connective tissue on the external surface of which several striated muscle fibers are intimately attached. The glandular follicles are enormously dilated and cystic, being distended by eosinophilic homogeneous colloid. Some follicles beneath the capsule appear compressed and distorted. The lining epithelium is everywhere compressed, the cells being very much flattened, so that it appears as a thin line between distended follicles. There are no papillomatous spurs, and no distinct evidence of parenchymal proliferation. The cell nuclei are small and very basophilic. Except for occasional thick connective tissue septa, no interfollicular substance is noted. A few small compressed blood vessels are present in the capsule and some of the septa.

The anatomic diagnosis of the specimen is *congenital colloid goiter with macrofollicular cystic degeneration.* The intrauterine death of the fetus was probably due to compression of the carotid arteries by the huge thyroid lobes (12).

Autopsy on the Dam.

About 16 months after this pregnancy the dam became blind and weak and was destroyed. At necropsy extensive acute and chronic infection of the pulmonary and intestinal tracts and of the meninges and uterus was found. The thyroid gland was moderately enlarged, the right lobe measuring 15× 7×2.5 centimeters, and the left lobe 11×1.5×2.5 centimeters. Upon examination the gland is uniformly brownish-red in color, and on section it appears fleshy. There are no adenomatous nodules. Histologically striking hyperplasia of the parenchymal element is observed. The epithelial cells are large, being high cuboidal in shape. The nuclei, too, are large and vesicular. The colloid content is considerably less than normal.

Discussion.

Simple, non-toxic goiter is found in a variety of animals, not only in adults, but in the newborn as well (2, 3, 4, 6, 7, 13). In fact, according to Marine (8), goiter may occur in any land or fresh water animal. It is said that wild animals never develop goiter even in regions where endemic goiter is prevalent (14). However, Fox (3) has noted the affliction in captive wild animals at the Philadelphia Zoological Garden.

In acquired goiter, human as well as animal, a definite goiter cycle has been established wherein the hyperactive phase is associated with glandular hyperplasia. Colloid goiter represents a resting state in the cycle, a physiologic return to normal which is expressed anatomically by an accumulation of colloid (7, 8). Joest (4) cites a case described by Johne, of a colloid struma in an adult dromedary.

In congenital goiter, which per se is not infrequent, by far the usual pathologic picture is one of hyperactivity—parenchymal hyperplasia with very little colloid (2, 4, 7, 10, 12, 13, 14). Marine (8) explains this as a physiologic reaction in the fetal gland compensating for the increased demands on the maternal thyroid that often obtain during pregnancy. This causal relationship is well demonstrated in the experimental production of congenital goiter by almost completely extirpating the maternal thyroid (2, 9). Abbott & Ball (1) in a study of 100 fetal and newborn thyroid glands state that it is reasonable to assume that the pathologic changes in the fetal thyroid are induced by the same type of stimulus that exists in the adult.

If the etiology of hyperplastic parenchymatous struma in the newborn is reasonably clear, the pathogenesis of a congenital colloid goiter is equally unclear. The latter is uncommon, the cystic form being extremely rare (4, 10, 12). In most cases of the colloid type there is a coexisting

hyperplasia. This, however, may be obscured by the colloid, so that one may be unable to affirm or deny its presence (11, 12). Since a paucity of colloid is a striking feature of the normal thyroid in the newborn, the association of hyperplasia with colloid is significant. It lends color to the concept that congenital colloid goiter originates in a hyperplastic thyroid rather than that it develops de novo.

In the case presented here, the enormous cystic dilatation of the follicles would readily obfuscate histologic evidence of preceding hyperplasia. It appears likely that the maternal thyroid was already in the hyperactive phase during the period of pregnancy, and initiated the fetal changes which went on to regression. One cannot say from this case alone whether a dysontogenetic hypersecretion of colloid ensued or whether some other factors came into play to produce the final picture.

Congenital goiter in the one-humped camel is not unknown. Leese (5) in a treatise on this species writes that the thyroid glands may be enormously enlarged at birth, and that death from suffocation may occur shortly after delivery. At times the goiter may persist without change throughout life, or may even become larger, reaching the size of a man's head and interfere with grazing from the ground. While Leese does not describe the anatomic type of goiter, such large masses would seem to be similar to the cystic colloid struma of this fetus.

Leese emphasizes the occurence of congenital goiter among camels out of dams confined to zoos or to ships during long sea-voyages, and suggests that insufficient exercise of the pregnant dam may be a predisposing factor. However, we now know that goiter is due to a relative or absolute lack of iodine which leads to a work hypertrophy of the thyroid (Marine (8)). The mediate factors involved include various dietary faults and the increased metabolic requirements in pregnancy and febrile, toxic states, any or all of which may be present in captive animals.

Summary.

1. A case is reported of a congenital macrofollicular cystic colloid goiter occurring in a full-term, still-born dromedary. The goiter resulted in dystocia.

2. Such a struma is rare not only in the lower animals but in humans as well.

3. An autopsy performed on the dam 16 months after delivery disclosed marked parenchymal hyperplasia of the thyroid together with severe acute and chronic infection of the pulmonary and intestinal tracts, meninges and uterus.

4. It is likely that the maternal thyroid was already hyperplastic during pregnancy, and initiated the fetal pathology. The cystic changes in the fetal gland would readily obscure evidence of parenchymatous hyperplasia—the usual picture in congenital goiter.

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EXPLANATION OF THE PLATE.

Plate I.

Cut surface of the fetal thyroid gland in a case of congenital macrofollicular cystic colloid goiter. Note the uniform enlargement, the glistening, translucent appearance, and the coarsely honeycombed structure. No normal thyroid tissue present. Each lobe measures 25 × 15 × 8 centimeters. (Photographed under water).